An Epidemiological Perspective of Obsessive-Compulsive Disorder in Children and Adolescents

*Joshua Fogel, Ph.D.
Affiliations:Johns Hopkins University, Bloomberg School of Public Health, Department of Mental Health[1]; Department of Biostatistics[2]; School of Medicine, Department of Psychiatry and Behavioral Sciences[3]

Abstract

Obsessive-compulsive disorder (OCD) is reviewed from an epidemiological perspective. OCD is defined according to the DSM-IV and ICD-10, with differences noted between these two classification systems. The epidemiological rubrics of quantity (prevalence), location (genetic methods and gender differences), cause (genetic etiology), and causal mechanisms (natural history and clinical course) are reviewed. The review concludes that more research is needed to further understand the epidemiology of OCD in children and adolescents, both from a Canadian and worldwide perspective.

Introduction

Obsessions are defined as "persistent ideas, thoughts, impulses, or images that are experienced as intrusive and inappropriate and that cause marked anxiety or distress" (American Psychiatric Association, 1994). Compulsions are defined as "repetitive behaviors or mental acts the goal of which is to prevent or reduce anxiety or distress, not to provide pleasure or gratification" (American Psychiatric Association, 1994). Associated features of OCD include avoiding situations that relate to the obsessional content, hypochondriacal worries, guilt, a strong sense of responsibility for the symptoms, and sleep difficulties. Compulsion performance can become all consuming and result in serious marital, occupational, and/or social disability (American Psychiatric Association, 1994).

The Diagnostic and Statistical Manual of Mental Disorders - 4th edition (DSM-IV; American Psychiatric Association, 1994) and International Statistical Classification of Diseases and Related Health Problems - 10th Revision (ICD-10; World Health Organization, 1992) are the two most commonly used manuals for conceptualizing psychiatric disorders. The DSM-IV is the manual typically used in the United States/Canada while the ICD-10 is more commonly used in Europe. Table 1 describes classification of OCD according to both manuals.

A DSM-IV diagnosis of OCD is made when all five criteria above are met. Note that category "A" can be either obsessions or compulsions. Also category "B" is not required for a diagnosis of OCD in children. Category "C" differentiates between one having delusions where one does not realize the obsessions and/or compulsions are unreasonable. If an individual for most of the time does not recognize that the obsessions and/or compulsions are excessive or unreasonable, one specifies "with poor insight". This specification can be used for an individual on the boundary between delusions and OCD. Even with this specification, the individual qualifies for a diagnosis of OCD. Also, DSM-IV classifies OCD as an anxiety disorder while ICD-10 does not.

One Austrian study comparing a clinical sample of children and adolescents with OCD showed poor agreement between the two classification symptoms. These authors concluded that DSM-IV criteria are preferred over the ICD-10 as they allow for a more detailed and age-independent description of the symptoms of OCD (Steinberger & Schuch, 2002). A recommendation for clinicians is to be aware of the diagnostic criteria used in the studies that they read as different classification systems may show different results.

Epidemiological Rubrics

Quantity

Different studies from different countries and populations are reviewed below. In a study in the southeastern US (Valleni-Basile et al., 1996) of students from grade 7 to 9, 0.7% one-year incidence of OCD and 8.4% one-year incidence of subclinical OCD is reported. They also quote studies of OCD in adolescents with community prevalence of 0.2-1.2%.

Apter et al. (1996) studying 16-17 year old Israeli army inductees determine with DSM-IV criteria a lifetime prevalence of 2.3% for OCD and 3.9% for subclinical OCD. They quote prevalence studies showing for high school students 0.35% with OCD and 2% with subclinical OCD. They quote another Israeli army inductee study showing 3.6% point prevalence and a non-Israeli study showing 3.0% point prevalence of OCD in adolescents. A New Zealand study of 18 year olds found 4.0% prevalence (Zohar, 1999).

Also, to the author's knowledge, only one epidemiological study reports the prevalence of obsessive-compulsive symptoms in children where 2.8-4.5% ranges are reported for a German sample of 8-year olds (Thomsen, 2000). No Canadian epidemiological studies exist that study the prevalence of OCD in children and adolescents. Weissman et al. (1994) pooled the data from 7 nations/regions worldwide and reports data from an adult Canadian epidemiological study done in Edmonton that show 1.4% annual prevalence and 2.3% lifetime prevalence of OCD. The DSM-IV quotes lifetime prevalence of 2.5% and one-year prevalence of 1.5-2.1% for OCD but does not categorize these prevalence rates

*Correspondence: Joshua Fogel, Ph.D.
Johns Hopkins University, Bloomberg School of Public Health, 624 North Broadway, Suite 861, Baltimore, MD 21205
e-mail: jfogel@jhsph.edu

for children, adolescents, and adults. Overall these studies can be summarized to show that OCD ranges for adolescents from 0.35-4.0%.

Location

Genetic studies do not report separate categories for children and adolescents and the genetic studies are reviewed below. Family studies using the family history method (indirect interview) show a rate of 32% for obsessive traits in first-degree relatives of OCD patients. Another study showed that 15.9% of parents are affected with OCD traits. Using the family study method (direct interview) of probands and families, 13% of all first-degree relatives had OCD. In a Mexican study, 4.9% first-degree relatives had OCD (Wolff, Alsobrook, & Pauls, 2000).

Also, the rate of OCD among first-degree relatives of Tourette's syndrome probands was significantly higher than rates of those from the general population (interview method unknown). Also, some forms of OCD are related to Tourette's syndrome, some are familial, and the inheritance of OCD is consistent with the transmission of an autosomal dominant genetic locus (Horwath & Weissman, 2000).

Genetic research shows no difference between male and female probands or between affected male and female parents for age of onset (Cavallini, Albertazzi, Bianchi, & Bellodi, 2002). These results are interesting in that the patients sampled were from a clinical sample and other clinical non-genetic studies show no gender differences. However, population based approaches show gender differences as shown from the ECA study (e.g., see Horwath & Weissman, 2000; Karno et al., 1988).

With regard to gender differences, there again is limited research with regard to children and adolescents. One US study showed higher prevalence of 3.3% for adolescent males as compared to 2.6% for females (Zohar, 1999) while an Israeli study of army inductees showed no gender differences between males and females in lifetime prevalence and also endorsement of a variety of OCD symptoms (Apter et al., 1996). The one reported Canadian study done in Edmonton shows lifetime prevalence of 2.7% for females and 2.0% for males (Weissman et al., 1994). In general, among adult studies, epidemiological samples show gender differences, while clinical patient samples do not (Horwath & Weissman, 2000; Weissman et al., 1994).

Cause

It is difficult to find a true cause, as there are many criteria involved before one can infer causation. Almost all of the research quoted below is from adult populations, which can be helpful for understanding the child and adolescent populations. Suspected causal influences for OCD include cocaine use, female gender, not working for pay, a prior history of alcohol dependence, affective disorder, and phobic disorder (Crum & Anthony, 1993). Other possible suspected causal influences reported for OCD include birth abnormalities, heritability, temper tantrums, intelligence, neuropsychological status, parental mental health, socioeconomic status, language impairment, Tourette's syndrome, and eating disorders (Douglass et al., 1995). Douglass et al. in a New Zealand sample conclude that perinatal problems/complications, parental educational status, maternal IQ, parental SES, a variety of habits/behaviors, IQ, neuropsychological tests, eating disorders, and Tourette's syndrome are not causal influences for OCD while depression in early adolescence and possibly substance dependence are suspected causal influences for OCD (Douglass et al., 1995). Religion and birth order are not causal influences for OCD (Nestadt et al. 1998). Black race, increasing age, more baseline undesirable life events and fewer desirable life events, and medium to high SES are suspected causal influences for OCD (Valleni-Basile et al., 1996). Also, there are higher odds ratios for those with OCD than those without OCD for lifetime major depression and also other anxiety disorders (Weissman et al. 1994). In a New Zealand sample, OCD was comorbid with major depression, social phobia, and substance dependence (Douglass et al., 1995). Overall, these suspected causal influences are numerous, yet they have not been shown/replicated in many studies. Replications and further studies are necessary in order to determine the suspected causal influences for OCD. At present, it is difficult to conclude the specific suspected causal influences for OCD.

As stated above, the pursuit of causation is challenging. Genetic approaches are a way to suggest causation. They include twin studies, segregation analysis, linkage analysis, and association studies.

Twin studies show that 63% of monozygotic twins are concordant for OCD. Also, in a Japanese study there was 20% concordance in 4 pairs of dizygotic twins and 80% concordance in 10 pairs of monozygotic twins (Wolff et al., 2000). In a sample of 1,054 twins, using principal components analysis of the Padua Inventory of Obsessions and Compulsions the authors conclude that obsessions have 33% heritability and compulsions, a 26% heritability (Micallef & Blin, 2001). These studies show that there is a large genetic influence to OCD. However, there are environmental factors for OCD as there was not 100% concordance for the monozygotic twins.

Segregation analysis studies of OCD are not as conclusive as the other methods. One study of OCD probands determined that one could not reject autosomal dominant or recessive models. Another study of families with OCD probands showed contribution of a major gene, but they could not determine a specific genetic model of dominant, recessive, or additive. Overall we can conclude that genes (and/or multiple genes) contribute to OCD but it is difficult to exactly model it (Wolff et al., 2000).

Linkage analysis studies show negative results for genetic causes. One study performing linkage analysis using a three-generation family having OCD and tics, did not have a statistically significant log odds ratio for the 4p13 region (Wolff et al., 2000). Another study had negative results on a battery of genes for receptors and enzymes involved in both dopamine (O4 receptor; dopamine transporter) and serotonin (5-HT, 1Dbeta; serotonin 2A receptor) neurotransmitters of OCD (Micallef & Blin, 2001; Wolff et al., 2000).

Association studies show no association between DRD3 and OCD, DRD4 and OCD, and no alteration in coding region for SLC6A4 and OCD. However, there is an association between the long form of the promoter SLC6A4 and OCD, for the COMT gene, there is a L/L genotype significance with OCD, and there is a sexually dimorphic association between OCD and the allele of the MAOA gene (Wolff et al., 2000).

Causal Mechanisms

Epidemiological mechanisms are based on natural history studies and clinical course studies. Natural history studies show that OCD has clinical manifestations that change as time progresses; the number of OCD symptoms gradually increases during childhood and then decreases during late adolescence and early adulthood. OCD is more severe in males where OCD developed before age 10 and in females where OCD developed after age 10. It is controversial if those with subclinical symptoms of OCD progress to OCD. The debated issue is if subclinical OCD is a stage of development or a level of severity. Also, children and adolescents can change from one category of OCD to another (Mataix-Cole et al., 2002). However, there is a pattern of moving from more severe to less severe OCD categories (Valleni-Basile et al., 1996).

Clinical course studies show that in children there is a positive association of OCD with increased IQ throughout adolescence. As they aged, adolescents with OCD and tics were more likely to have tics in adult years. Also, childhood conduct disturbance predicted early adolescent OCD. Childhood tics and early adolescent separation anxiety predicted the development of OCD symptoms in late adolescence. Tics and ADHD in late adolescence predicted more OCD symptoms in adult years (Peterson, Pine, Cohen & Brook, 2001).

Genetic studies of symptoms show that the median age at onset in the offspring generation was lower than the parent generation. This suggests an "anticipation effect" where there is a reduced age at onset for a specific disease (i.e., OCD) in offspring generations (Cavallini et al., 2002). Family aggregation exists in OCD but the evidence is not as strong as that shown for a genetic anticipation effect and family aggregation is helpful but only a link in the chain to suggest true causation.

Future Epidemiological Research

Many areas exist for future research. Studies are needed to determine the incidence and prevalence of OCD in children. Canadian studies are needed to determine the possibly unique characteristics of a Canadian sample of children and adolescents. Also ethnic Canadian groups should be studied to see if there are different rates for the Anglophone, Francophone, and First Nation populations.

Suspected causal influences for OCD are often a hodgepodge of studies with conflicting results and more research is needed to refine them. Another area relates to the differences observed between clinical and epidemiological studies and future research should try to resolve some of these conflicting results. In conclusion, OCD has a number of future research questions that may be asked to help further understand it. With further studies we may be able to understand these currently unanswered questions, which will help expand our knowledge of OCD.

References

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders - 4th edition. Washington, DC: Author.

Apter A, Fallon TJ Jr, King RA, Ratzoni G, Zohar AH, Binder M, Weizman A, Leckman JF, Pauls DL, Kron S, & Cohen DJ. (1996). Obsessive-compulsive characteristics: from symptoms to syndrome. Journal of the American Academy of Child & Adolescent Psychiatry, 35(7), 907-912.

Cavallini MC, Albertazzi M, Bianchi L, & Bellodi L. (2002). Anticipation of age at onset of obsessive-compulsive spectrum disorders in patients with obsessive-compulsive disorder. Psychiatry Research, 111(1), 1-9.

Crum RM, & Anthony JC. (1993). Cocaine use and other suspected risk factors for obsessive-compulsive disorder: a prospective study with data from the Epidemiologic Catchment Area surveys. Drug and Alcohol Dependence, 31(3), 281-295.

Douglass HM, Moffitt TE, Dar R, McGee R, & Silva P. (1995). Obsessive-compulsive disorder in a birth cohort of 18-year-olds: prevalence and predictors. Journal of the American Academy of Child & Adolescent Psychiatry, 34(11), 1424-1431.

Horwath E, & Weissman MM. (2000). The epidemiology and cross-national presentation of obsessive- compulsive disorder. Psychiatric Clinics of North America, 23(3), 493-507.

Karno M, Golding JM, Sorenson SB, & Burnam MA. (1988). The epidemiology of obsessive-compulsive disorder in five US communities. Archives of General Psychiatry, 45(12), 1094-1099.

Mataix-Cols D, Rauch SL, Baer L, Eisen JL, Shera DM, Goodman WK, Rasmussen SA, & Jenike MA. (2002). Symptom stability in adult obsessive-compulsive disorder: data from a naturalistic two-year follow-up study. American Journal of Psychiatry, 159(2), 263-268.

Micallef J, & Blin O. (2001). Neurobiology and clinical pharmacology of obsessive-compulsive disorder. Clinical Neuropharmacology, 24(4), 191-207.

Nestadt G, Bienvenu OJ, Cai G, Samuels J, & Eaton WW. (1998). Incidence of obsessive-compulsive disorder in adults. Journal of Nervous and Mental Disease, 186(7), 401-406.

Peterson BS, Pine DS, Cohen P, & Brook JS. (2001). Prospective, longitudinal study of tic, obsessive-compulsive, and attention-deficit/hyperactivity disorders in an epidemiological sample. Journal of the American Academy of Child & Adolescent Psychiatry, 40(6), 685-695.

Steinberger K, & Schuch B. (2002). Classification of obsessive-compulsive disorder in childhood and adolescence. Acta Psychiatrica Scandinavica, 106, 97-102.

Thomsen PH. (2000). Obsessions: The impact and treatment of obsessive-compulsive disorder in children and adolescents. Journal of Psychopharmacology, 14(2), S31-S37.

Valleni-Basile LA, Garrison CZ, Waller JL, Addy CL, McKeown RE, Jackson KL, & Cuffe SP. (1996). Incidence of obsessive-compulsive disorder in a community sample of young adolescents. Journal of the American Academy of Child & Adolescent Psychiatry, 35(7), 898-906.

Weissman MM, Bland RC, Canino GJ, Greenwald S, Hwu HG, Lee CK, Newman SC, Oakley-Browne MA, Rubio-Stipec M, Wickramaratne PJ, & et al. (1994). The cross national epidemiology of obsessive compulsive disorder. The Cross National Collaborative Group. Journal of Clinical Psychiatry, 55, S5-S10.

Wolff M, Alsobrook JP 2nd, & Pauls DL. (2000). Genetic aspects of obsessive-compulsive disorder. Psychiatric Clinics of North America, 23(3), 535-544.

World Health Organization (1992). International Statistical Classification of Diseases and Related Health Problems - 10th Revision. Geneva: Author.

Zohar AH. (1999). The epidemiology of obsessive-compulsive disorder in children and adolescents. Child and Adolescent Psychiatric Clinics of North America, 8(3), 445-460.

Table 1

Classification of Obsessive-Compulsive Disorder

Categories	DSM-IV	ICD-10
A. Presence of recurrent obsessions and/or compulsions	yes	yes
B. Patient realizes they are excessive and unreasonable (not required for children)	yes	yes, but no mention of the children exclusion
C. Severe enough to be time consuming or cause marked distress or significant impairment	yes	yes, but no mention of time consuming
D. If other axis I disorder present, obsessional or compulsive content not restricted to it	yes	mentions comorbidity of depression and OCD – OCD diagnosis preferred only in absence of depressive episode
E. Disturbance not due to direct physiological effects of substance or general medical condition	yes	not mentioned